Complete Summary

GUIDELINE TITLE

Management of cocaine-associated chest pain and myocardial infarction. A scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology.

BIBLIOGRAPHIC SOURCE(S)

McCord J, Jneid H, Hollander JE, de Lemos JA, Cercek B, Hsue P, Gibler WB, Ohman EM, Drew B, Philippides G, Newby LK, American Heart Association Acute Cardiac Care Committee of the Council on. Management of cocaine-associated chest pain and myocardial infarction: a scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation 2008 Apr 8;117(14):1897-907. [113 references] PubMed

GUIDELINE STATUS

This is the current release of the guideline.

COMPLETE SUMMARY CONTENT

SCOPE

METHODOLOGY - including Rating Scheme and Cost Analysis RECOMMENDATIONS EVIDENCE SUPPORTING THE RECOMMENDATIONS BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS CONTRAINDICATIONS IMPLEMENTATION OF THE GUIDELINE INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT

CATEGORIES IDENTIFYING INFORMATION AND AVAILABILITY

DISCLAIMER

SCOPE

DISEASE/CONDITION(S)

Cocaine-associated chest pain and myocardial infarction

GUIDELINE CATEGORY

Diagnosis Management Prevention **Treatment**

CLINICAL SPECIALTY

Cardiology Critical Care Emergency Medicine Family Practice Internal Medicine

INTENDED USERS

Physicians

GUIDELINE OBJECTIVE(S)

To provide a critical review of the literature on cocaine-associated chest pain and myocardial infarction (MI) and to give guidance for diagnostic and therapeutic interventions

TARGET POPULATION

Patients with suspected or known cocaine-associated chest pain and myocardial infarction

INTERVENTIONS AND PRACTICES CONSIDERED

Diagnosis

- 1. Self-report
- 2. Urinalysis
- 3. Electrocardiogram
- 4. Cardiac biomarkers
- 5. Myocardial perfusion imaging
 - Echocardiography
 - Coronary angiography
- 6. Evaluation in a chest pain unit

Management/Treatment

- 1. Initial management of cocaine-associated myocardial ischemia or infarction
 - Benzodiazepines
 - Aspirin
 - Nitroglycerin
 - Calcium channel blocker
 - Phentolamine
 - Beta-blockers
 - Labetalol
- 2. ST-segment myocardial infarction
 - Percutaneous coronary intervention (PCI)
 - Fibrinolytic therapy
 - Stents: bare metal, drug-eluting
 - Beta-blockers

- Nitroglycerin
- Calcium channel blockers
- Phentolamine
- Other therapeutic agents: antiplatelet, antithrombin, glycoprotein IIb/IIIa antagonists, clopidogrel, unfractionated heparin, lowmolecular-weight heparin, direct thrombin inhibitors
- 3. Ventricular tachyarrhythmias
 - Sodium bicarbonate
 - Lidocaine
- 4. Discharge management and secondary prevention
 - Psychosocial interventions for cessation of cocaine use
 - Risk factor modification
 - Medical therapy for myocardial infarction (MI) or atherosclerosis
 - Individualized decision on future beta-blocker usage

MAJOR OUTCOMES CONSIDERED

- Myocardial infarction, fatal and nonfatal
- Cardiac arrest
- Recurrent ischemic events
- Continued cocaine use
- Mortality

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The Writing Committee conducted a comprehensive search of the medical literature concerning cocaine-associated chest pain and myocardial infarction (MI). The literature search included English-language publications on humans and animals from 1960 to 2007. In addition to broad-based searching concerning cocaine, specific targeted searches were performed on cocaine and the following topics: myocardial infarction, chest pain, emergency department (ED), aspirin, nitroglycerin, calcium channel blocker, benzodiazepine, thrombolytics, phentolamine, heparin, primary angioplasty, electrocardiogram (ECG), and stress testing. Literature citations were generally limited to published articles listed in Index Medicus.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Levels of Evidence

Level of Evidence A: Data derived from multiple randomized clinical trials.

Level of Evidence B: Data derived from a single randomized trial or nonrandomized studies.

Level of Evidence C: Only consensus opinion of experts, case studies, or standard of care.

METHODS USED TO ANALYZE THE EVIDENCE

Systematic Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Classification of Recommendations

Class I: Conditions for which there is evidence for and/or general agreement that the procedure or treatment is beneficial, useful, and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment.

Class IIa: The weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

This statement was approved by the American Heart Association (AHA) Science Advisory and Coordinating Committee on December 20, 2007.

Expert peer review of AHA Scientific Statements is conducted at the AHA National Center.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Diagnostic Strategies

Establishing cocaine use in a patient presenting with chest pain should depend primarily on self-reporting. As the use of cocaine may influence treatment strategies, patients being evaluated for possible acute coronary syndrome (ACS) should be queried about the use of cocaine; this especially applies to younger patients. Not enough information exists to definitely recommend the routine screening of particular subgroups of patients. The qualitative determination of cocaine metabolites in the urine should be done only in specific cases, including when the patient is unable to communicate and no other reliable source of the history is available. When confronted with patients with no or few risk factors for coronary artery disease presenting with myocardial infarction (MI), especially those who are young or have a history of illicit drug use; however, measuring cocaine urine metabolites may be prudent. The evaluation of cocaine-associated chest pain in the emergency department (ED) is in general the same as evaluation of patients for possible ACS without cocaine use: electrocardiogram (ECG), serial cardiac markers, and some form of stress testing.

Electrocardiogram

An abnormal ECG has been reported in 56% to 84% of patients with cocaine-associated chest pain; however, many of these patients are young and commonly have the normal variant of early repolarization, which may be interpreted by physicians as an abnormal ECG finding.

Cardiac Biomarkers

Cocaine ingestion may cause rhabdomyolysis with consequent elevation in myoglobin and total creatine kinase levels, which may confound the diagnosis of cocaine-associated MI. Cardiac troponins are the most sensitive and specific

markers for the diagnosis of cocaine-associated MI; therefore, their use is preferred in patients with possible ACS in the setting of cocaine use.

Myocardial Perfusion Imaging

Echocardiography

Compared with nonusers, long-term cocaine users have a higher left ventricular mass index (mean 103 ± 24 g/m² among users compared with 77 ± 14 g/m² in nonusers) and thickness of the posterior wall (>1.2 cm in 44% of users compared with 11% in nonusers).

Echocardiography also yields information concerning systolic and diastolic function and valvular structure that may affect treatment strategies.

The appropriate diagnostic evaluation for these patients remains unclear. Practitioners should follow general principles for risk stratification of patients with possible ACS. In light of the underlying electrocardiographic abnormalities, if a stress test is ordered, most patients would benefit from stress testing with imaging, either echocardiography or nuclear.

Evaluation in a Chest Pain Unit

Studies suggest that risk stratification on the basis of well-established criteria, including electrocardiogram (ECG) changes and positive cardiac troponin, is feasible and safe in patients with chest pain associated with cocaine use. Patients at high risk should be admitted to monitored beds.

In the absence of ischemic electrocardiographic changes or positive cardiac markers, intermediate- and low-risk patients can be safely managed in a chest pain observation unit for 9 to 12 hours, which can obviate the need for hospital admission in the majority of these patients.

The guideline developers recommend that stress testing be optional for patients with cocaine-associated chest pain who have had an uneventful 9 to 12 hours of observation. Stress testing can be performed at the time of observation or on an outpatient basis and should be considered depending on cardiac risk factors and ongoing symptoms.

Therapeutic Strategies

General Considerations

Patients with cocaine-associated chest pain, unstable angina, or MI should be treated similarly to those with traditional ACS or possible ACS, with some notable exceptions (see Figure in original guideline document).

Therapeutic recommendations are based on animal studies, cardiac catheterization studies, observational studies, case series, and case reports (see Table below). Unlike patients with ACS unrelated to cocaine use, cocaine users should be provided with intravenous benzodiazepines as early management. In

the setting of cocaine use, benzodiazepines relieve chest pain and have beneficial cardiac hemodynamic effects. The neuropsychiatric symptoms and cardiovascular complications of cocaine use are interrelated; therefore, management of neuropsychiatric manifestations favorably impacts the systemic manifestations of cocaine toxicity.

Table. Scientific Strength for Treatment Recommendations for Initial Management of Cocaine-Associated Myocardial Ischemia or Infarction

Therapy	Classification of Recommendation/Level of Evidence	Controlled Clinical Trials	Cardiac Catheterization Laboratory Studies	Case Series or Observational Studies	Cas Repo	
Benzodiazepines	I/B	Х			X	
Aspirin	I/C			X		
Nitroglycerin	I/B	Х	X	X		
Calcium Channel Blocker	IIb/C		X			
Phentolamine	IIb/C		X		X	
β-Blockers	III/C		X		X	
Labetalol	III/C		X		X	

No. of patients in studies/reports: benzodiazepines, 67; nitroglycerin, 67; phentolamine, 45; calcium channel blocker, 15; beta-blockers without alpha-blocking properties, 30; labetalol, 15; and fibrinolytics, 66.

Hypertension and tachycardia may not require direct treatment. In a patient with definite ACS, these signs need to be addressed. In a patient with chest pain of unclear origin, hypertension and tachycardia should be treated conservatively. Resolution of anxiety with a benzodiazepine will often lead to resolution of the hypertension and tachycardia. When sedation is not successful, hypertension can be managed with sodium nitroprusside, nitroglycerin, or intravenous phentolamine.

ST-Segment-Elevation Myocardial Infarction

Timely percutaneous coronary intervention by experienced operators in high-volume centers is preferred over fibrinolytics in ST-segment-elevation MI and is even more desirable in the setting of cocaine use. Fibrinolytic therapy should be reserved for patients who are clearly having an ST-segment-elevation myocardial infarction (MI) who cannot receive direct percutaneous coronary intervention.

Patients with ongoing cocaine abuse may have poor compliance with the longterm antiplatelet regimen of aspirin and clopidogrel, potentially increasing their risk for subacute and late thrombosis. Therefore, the guideline developers recommend very careful consideration of the probability of long-term compliance before a drug-eluting stent is used in a patient with cocaine-associated MI. In most cases, a bare metal stent would be preferable. Patients with non-ST-elevation MI or unstable angina are at higher risk for subsequent events and may benefit from an early invasive approach with cardiac catheterization and revascularization, just as patients with ACS unrelated to cocaine do.

Beta-Blockers

Although beta-blocker administration is recommended for patients with MI unrelated to cocaine because it can lead to lower mortality rates, deaths from cocaine-associated MI are exceedingly low, altering the risk-benefit ratio.

The use of beta-adrenergic antagonists for the treatment of cocaine toxicity should be avoided in the acute setting.

At discharge, beta-blockers should be considered for patients with coronary artery disease or left ventricular dysfunction in certain situations (see below for Discharge Management and Secondary Prevention).

Nitroglycerin

One case series and 2 randomized controlled trials have shown that nitroglycerin relieves cocaine-associated chest pain. Nitroglycerin is similar to benzodiazepines with respect to the relief of cocaine-associated chest pain. Cardiac catheterization studies demonstrate that nitroglycerin reverses cocaine-associated vasoconstriction. Nitroglycerin can also be used to control hypertension when a patient does not respond to benzodiazepines.

Calcium Channel Blockers

Calcium channel blockers should not be used as a first-line treatment but may be considered for patients who do not respond to benzodiazepines and nitroglycerin.

Phentolamine

There are anecdotal reports about the safety and efficacy of phentolamine, an alpha-antagonist, for the treatment of cocaine-associated ACS. The administration of phentolamine returned coronary arterial diameter to baseline, suggesting that phentolamine may be useful for the treatment of cocaine-associated ischemia.

Other Therapeutic Agents

The guideline developers recommend aspirin be routinely administered and unfractionated heparin or low-molecular-weight heparin be given to patients with cocaine-associated MI unless there is a contraindication. Aspirin has been shown to be safe when used in an observation unit in patients with cocaine-associated chest pain.

Ventricular Tachyarrhythmias

The treatment of ventricular arrhythmias depends on the time interval between cocaine use, arrhythmia onset, and treatment. Ventricular arrhythmias occurring immediately after cocaine use result from the local anesthetic (sodium channel) effects on the myocardium. These arrhythmias may respond to the administration of sodium bicarbonate, similar to arrhythmias associated with other type IA and type IC agents. In addition, one animal model suggested that lidocaine exacerbated cocaine-associated seizures and arrhythmias as a result of similar effects on sodium channels; however, this finding has not been confirmed in other animal models. Bicarbonate therapy may be preferable and has been used effectively. Ventricular arrhythmias that occur several hours after the last use of cocaine are usually secondary to ischemia, the management of which should be the first goal for treatment. Standard management for ventricular arrhythmias, including lidocaine, is reasonable for persistent or recurrent ventricular arrhythmias. No data exist concerning the efficacy of amiodarone in clinical cocaine intoxication.

Discharge Management and Secondary Prevention

Cessation of cocaine use should be the primary goal of secondary prevention. Several options for psychosocial intervention exist, including individual and group counseling, psychotherapy, and cognitive therapy. Preliminary data suggest that a combination of intensive group and individual drug counseling has the greatest impact on recurrent cocaine use.

Aggressive modification of traditional risk factors is indicated for patients with MI or with evidence of atherosclerosis. This includes smoking cessation, hypertension control, diabetes control, and aggressive lipid-lowering therapy with a target low-density lipoprotein level <70 mg/dL. Although these strategies have not been tested specifically for patients who use cocaine, they are standard for patients with underlying coronary artery disease.

Patients with evidence of MI or atherosclerosis should receive long-term antiplatelet therapy with aspirin. In addition to aspirin, clopidogrel should be given for at least 1 month to patients who undergo percutaneous coronary intervention with bare metal stents and for at least 1 year for those treated with drug-eluting stents. Among patients treated medically (i.e., without percutaneous coronary intervention), the combination of antiplatelet therapy with aspirin and clopidogrel is clearly of benefit among patients with unstable angina and non–ST-segment–elevation MI not precipitated by cocaine use, but this regimen has not been studied in patients with cocaine-associated chest pain and MI. Selective use of the combination of aspirin and clopidogrel may be considered for those patients with cocaine-associated MI who have evidence of underlying coronary artery disease. Nitrates and calcium channel blockers may be administered to treat anginal symptoms but are not indicated for routine use. Angiotensin-converting enzyme inhibitors should be used in patients with left ventricular systolic dysfunction.

As noted above, beta-adrenergic antagonists should not be administered acutely in patients with cocaine-associated chest pain and/or MI because of concerns about provoking or exacerbating coronary spasm. Post-discharge use of beta-blockers, although clearly beneficial among patients with previous MI and cardiomyopathy who do not abuse cocaine, merits special consideration in the setting of cocaine abuse. Because recidivism is high among patients with cocaine-

associated chest pain, chronic beta-blocker use should be reserved for those with the strongest indications, including those with documented MI, left ventricular systolic dysfunction, or ventricular arrhythmias, in whom the benefits may outweigh the risks even among patients at risk for recurrent use of cocaine. This decision should be individualized on the basis of careful risk—benefit assessment and after counseling the patient about the potential negative interactions between recurrent cocaine use and beta-blockade.

CLINICAL ALGORITHM(S)

A clinical algorithm on the therapeutic and diagnostic recommendations in cocaine-associated chest pain is provided in the original guideline document.

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of evidence supporting the recommendations is not specifically stated for each recommendation.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Accurate diagnosis and appropriate treatment of cocaine-associated chest pain and myocardial infarction (MI)

POTENTIAL HARMS

Fibrinolytic Therapy

Case reports document adverse outcomes, such as a higher rate of intracranial hemorrhage, after fibrinolytic administration in patients who use cocaine.

Beta-blockers

- Coronary artery vasoconstriction is exacerbated by the administration of propranolol. The unopposed alpha-adrenergic effect leads to worsening coronary vasoconstriction and increased blood pressure. Multiple experimental models have shown that beta-adrenergic antagonists lead to decreased coronary blood flow, increased seizure frequency, and increased mortality.
- The American College of Cardiology/American Heart Association (ACC/AHA) ST-segment-elevation myocardial infarction (MI) guidelines state, "Betablockers should not be administered to patients with ST-segment-elevation MI (STEMI) precipitated by cocaine use because of the risk of exacerbating coronary spasm."
- Labetalol increases the risk of seizure and death in animal models of cocaine toxicity and does not reverse coronary artery vasoconstriction in humans.

CONTRAINDICATIONS

CONTRAINDICATIONS

Beta-blockers

The 2005 American Heart Association (AHA) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care state "propranolol is contraindicated in cocaine overdose" and "propranolol is contraindicated for cocaine induced ACS."

Calcium Channel Blockers

Short-acting nifedipine should never be used, and verapamil or diltiazem should be avoided in patients with evidence of heart failure or left ventricular dysfunction.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

IMPLEMENTATION TOOLS

Clinical Algorithm

For information about <u>availability</u>, see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better Living with Illness

IOM DOMAIN

Effectiveness Patient-centeredness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

McCord J, Jneid H, Hollander JE, de Lemos JA, Cercek B, Hsue P, Gibler WB, Ohman EM, Drew B, Philippides G, Newby LK, American Heart Association Acute 11 of 16

Cardiac Care Committee of the Council on. Management of cocaine-associated chest pain and myocardial infarction: a scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation 2008 Apr 8;117(14):1897-907. [113 references] PubMed

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2008 Apr 8

GUIDELINE DEVELOPER(S)

American Heart Association - Professional Association

SOURCE(S) OF FUNDING

American Heart Association

GUIDELINE COMMITTEE

Acute Cardiac Care Committee

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Boa
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Philippides	Medical Center					

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives \$10,000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns \$10,000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest

†Significant

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Ot
Shenghan Lai	Johns Hopkins	None	None	None	None	None	None	No
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Murray M. Mittleman	Beth Israel Deaconess Medical Center	None	None	None	None	None	None	No

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit.

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: Available from the American Heart Association Web site.

Print copies: Available from the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596; Phone: 800-242-8721

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

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